Table 1:
Nominal values for DDREF and RBE from FGR 13.

Tissue	DDREF[3]	RBEA	High dose RBE[4]
Breast	1	10	10
Leukemia	2	1	0.5
All others	2	20	10

Even with this simpler model, a full-scale parameter uncertainty analysis is prohibitive because of the large number of cases to be considered and difficulties in assigning uncertainty distributions to some of the parameter values of Eq. 1. For each risk coefficient, a limited analysis based on propagation of uncertainties was performed to assess the sensitivity of predictions of Eq. 1 to dominant sources of uncertainty in each of the parameter values di, ai, Di, Ri, and DDREFi,. The uncertainties were propagated through assignment of continuous uncertainty distributions to each of the parameter values Ri, ai, di, and Di , and application of random simulation techniques to the model represented by Eq. 9 to generate a range of possible values of each risk coefficient. The 5% and 95% values from the generated range formed the basis for assigning a nominal uncertainty interval for each risk coefficient. This incorporated evaluation of subjective judgements derived from an expert elicitation (NRC-CEC 1998), previously published reports on uncertainties (such as NCRP 1997; EPA 1999), and additional subjective judgments of the authors.

Assignment of uncertainties to the values R_i (age- and gender-averaged risk model coefficients for high dose for tissues i=1,2,...) was based on recently published judgments of nine independent experts on the health effects of radiation (NRC-CEC 1998). Assignment of uncertainties to the alpha RBEs, a_i, and the dose and dose rate effectiveness factor, $DDREF_i$, were the same as in an EPA report on uncertainties from whole-body low-LET radiation (EPA 1999). Assigned uncertainties for the RBEs were based on ranges of values determined from experimental and epidemiological studies of the relative carcinogenic effects of low- and high-LET radiation data, as discussed in recent documents (NAS, 1988; NCRP, 1990; ICRP 1991; EPA, 1991; EPA 1999). Conclusions on DDREFs were based on subjective evaluations of evidence from animal, laboratory, and to a limited extent on epidemiological studies applied to competing dose-response models. Uncertainties for the parameter values R_i, a_i, and the $DDREF_i$ were assumed to be independent of the radionuclide and exposure mode.

Characterization of uncertainties in the tissue-specific dose estimates d_i and D_i (respectively, low- and high-LET dose estimates for tissues i=1,2...) was more difficult – these uncertainties depend strongly on the radionuclide as well as the exposure mode and this topic has rarely been addressed in the literature. As described later, uncertainties in the values d_i and D_i were judged from results of a separate sensitivity analysis in which the typically dominant components of the ICRP's biokinetic and dosimetric scheme were varied within plausible ranges of values.

[3] *For doses < 0.2 Gy*

[4] *For doses >0.2 Gy*

Assignment Of Uncertainties To Components Of The Simplified Model

Risk Model Coefficients For High Dose And Dose Rate

The U.S. Nuclear Regulatory Commission (NRC) and the Commission of European Communities (CEC) recently conducted a joint study aimed at characterizing the uncertainties in predictions of the consequences of accidental releases of radionuclides into the environment (NRC-CEC, 1997, 1998). As part of the exercise, the experts were asked to provide 5%, 50%, and 95% quantiles of subjective probability distributions for the total number of radiation-induced cancer deaths and for the numbers of tissue-specific cancer deaths over a lifetime in a typical population of 100 million persons, each receiving a whole body dose of 1 Gy low LET radiation at a uniform rate over 1 min. With minor exceptions, the tissues considered in the NRC-CEC study are the same as those addressed in this report. In the present analysis, the uncertainty in site-specific cancer mortality risk estimates for high-dose, low-LET radiation was based on the judgments of the NRC-CEC experts.

In our analysis, a set of lognormal distributions represented the uncertainties in estimates of site-specific cancer deaths following a high dose of radiation at a high dose rate. For each cancer site, a lognormal distribution was constructed to match the conclusions of a given expert. Parameters of the resulting lognormal distributions representing the uncertainty in the age- and gender-averaged risk model coefficients, R_i , for high dose and dose rate are given in Table 2.

Table 2:
Mean and Standard Deviation of Distributions Representing The Uncertainties In The Log Transformed Cancer Mortality Risk Coefficients (cancer deaths per person-Gy) for high dose and dose rates[5].

Tissue	Mean	Standard deviation
Bone	-7.90	1.50
Breast	-5.03	0.85
Colon	-4.90	0.96
Leukemia	-4.80	0.50
Liver	-7.08	1.49
Lung	-3.90	0.80
Stomach	-5.92	1.27
Skin	-8.09	1.34
Thyroid	-7.47	1.23
Residual[6]	-3.78	0.98

[5] *Distributions are based on judgments of nine experts on the health effects of radiation (NRC-CEC, 1997).*

[6] *As defined in NCR-CEC (1997)*

Tissue-specific DDREFs

In Federal Guidance Report No. 13, a DDREF of 2 was applied to all cancer sites except breast, for which a value of 1 was applied. The distributions used here for representing uncertainties in the DDREF are described in the EPA report on uncertainties from whole-body low-LET radiation (EPA 1999), and are based on the approach developed in NCRP Report No. 126. For most sites, we have adopted a distribution that is uniform from 1 to 2, and falls off exponentially for values greater than 2. The two parts of the distribution are normalized so that: (1) the probability density function is continuous and (2) the integrals of the uniform and exponential portions are each 0.5. Mathematically, this probability density for the DDREF, f(x), can then be written:

$$f(x) = 0.5 \qquad 1 \# x \# 2 \qquad (2a)$$
$$f(x) = 0.5\, e^{-(x-2)} \qquad x > 2$$

The probability density function for breast given in Eq. 2b is somewhat narrower to reflect linear dose response results observed in several study populations and the apparent invariance in risk with dose fractionation (Hrubec et al. 1989, NAS 1990, Howe 1992, Tokunaga et al. 1994).

$$f(x) = 2\, e^{2(1-x)} \quad (2b)$$

Tissue-specific RBEs

In the derivation of the risk coefficients tabulated in FGR 13, alpha RBEs of 1, 10, and 20 were applied to red marrow (leukemia), breast, and all other tissues, respectively. For this analysis, uncertainty distributions assigned to tissue-specific RBEs were the same as those described in the EPA report on uncertainties from whole-body low LET radiation (EPA 1999). For most tissues, a lognormal distribution with geometric mean equal to the square root of 50, and a 90% probability assigned to the interval 2.5 to 20 was used. For leukemia, the uncertainty in RBE is represented using a uniform distribution between 0 and 1.

Estimates Of Absorbed Dose

Assignment of uncertainty distributions to the radionuclide-specific parameter values d_i and D_i (respectively, low- and high-LET dose estimates for tissues i=1,2...) for internally deposited radionuclides is particularly difficult because these values are end products of complex calculations involving a collection of uncertain biokinetic and dosimetric models, parameters, and assumptions. Current biokinetic models for elements generally are not process models, and their parameter values often do not represent measurable quantities. Conversion from internally distributed activity to tissue doses involves the application of specific energies (*SE* values) for numerous pairs of target and source organs, and the uncertainty in a given *SE* value depends on the types and energies of emitted radiations. Even if the information were available to assign meaningful uncertainty distributions to all parameter values of all biokinetic and dosimetric models applied in this report, this would not be a feasible task due to the numerous cases considered.

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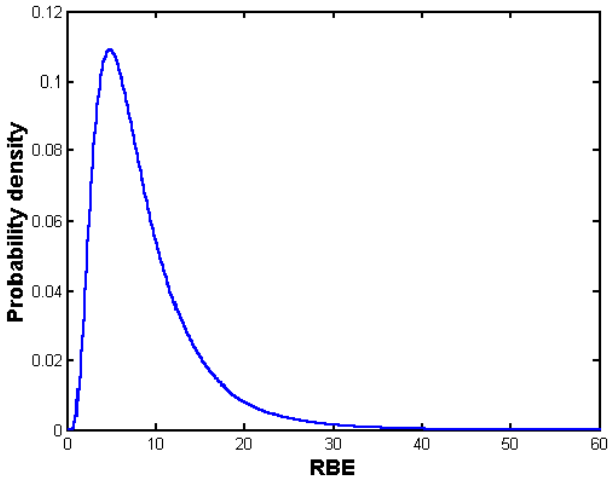
In view of such difficulties, a systematic scheme was devised to produce a Monte Carlo simulation of the absorbed doses. First, we created a data set of dose estimates that were calculated for a limited number of plausible alternatives of components that typically dominate the biokinetic and dosimetric models. The dominant components were identified using a relatively detailed sensitivity analyses for selected radionuclides. Then for each radionuclide addressed in this document, we constructed a few substantially different but plausible variants for each of those dominant components. The data set was based on a factorial design in which the absorbed dose estimates were calculated for each combination of the selected variants for the dominant components, with all other aspects of the biokinetic and dosimetric models left unchanged. Finally for each radionuclide, the data set was used to derive continuous distributions relating to each of the identified components from which doses were simulated.



The following components were judged to represent the dominant uncertainties in most situations: the rate of absorption from the respiratory tract to blood, the gastrointestinal absorption fraction (f_1 value), the systemic biokinetic model, and *SE* values for certain combinations of source and target organs and radiation types. For each radionuclide, we used 3 different values for f_1, 3 different systemic models, and 2 different values for *SE*. Thus for ingestion, at least 18 different sets of dose estimates corresponding to the 18 = 3×3×2 combinations of variations of the above components, were considered. For inhalation of a radionuclide of a given absorption type, at least 54 combinations were considered. Thus, the data set included at least 180 dose estimates for ingestion (for low LET radiation there are 18 estimates for each of 10 sites) per radionuclide and at least 540 dose estimates for inhalation. A portion of this data set is shown in Table 4, which shows dose estimates obtained using the ICRP value for *SE* for two of the ten tissue sites for ingestion of Ru-106.

For each radionuclide, there is an important difference between the variants selected for the systemic models and the variants selected for the other components. In general, the selected variants for the f_1 value, *SE* value, and rate of absorption from the respiratory tract were chosen with the aim to include a "low" and "high" value that encompass the range of most plausible values. This is much more difficult to do for systemic models, since for most radionuclides the universe of plausible models cannot be coherently defined using a single one-dimensional parameter. For each radionuclide, we assumed that the selected

systemic models used to construct the data set were randomly selected from the universe of all plausible systemic models. In contrast, we assumed the selected variants for f_1, *SE*, and the absorption rate from the respiratory tract represent quantile values from continuous distributions that represent expert subjective opinion on plausible values for these parameters. To illustrate the difference, suppose there is a radionuclide for which the f_1 and *SE* values are known, and for which the only uncertainties relate to the proper choice of a systemic model. The probability that the three selected systemic models (that form the basis for the data set) would all yield colon doses less than "true" colon dose would be $0.5^3 = 0.125$. In contrast, the selected f_1 values are the 5, 50, and 95% quantiles for the continuous distribution of plausible f_1 values. According to expert opinion, there would be only about a 5% chance that all three f_1 values are less than the true value.

For each radionuclide, doses were simulated separately for each of the systemic biokinetic models that were considered. This was accomplished by first estimating the functional relationship between the dose to each tissue and variables representing the other sources of variation (such as the f_1 value) using the data set of dose estimates. Then for each radionuclide, distributions were assigned to the gastrointestinal absorption fraction, standardized *SE* values, and in the case of inhalation absorption from the respiratory tract. Simulated doses to each tissue were then calculated by applying the estimated functional relationship to simulated values for the sources of variation (such as the f_1 value).

Table 3:
Colon and stomach dose estimates using the ICRP value for *SE* for Ru-106

Systemic Model	f1 -value	SE value	Colon Dose (Gy/Bq)	Stomach Dose (Gy/Bq)
first	0.005	ICRP	4.59E-08	1.69E-09
first	0.05 (ICRP)	ICRP	4.44E-08	1.71E-09
first	0.07	ICRP	4.37E-08	1.72E-09
ICRP	0.005	ICRP	4.60E-08	1.83E-09
ICRP	0.05 (ICRP)	ICRP	4.55E-08	3.13E-09
ICRP	0.07	ICRP	4.54E-08	3.71E-09
third	0.005	ICRP	4.59E-08	1.76E-09
third	0.05 (ICRP)	ICRP	4.49E-08	2.46E-09
third	0.07	ICRP	4.45E-08	2.77E-09

The final step of the simulation was to fully account for uncertainties in risks associated with choice of the systemic absorbed dose model. We plan to provide details on how this was accomplished in an EPA/ORNL technical report.

Results

Table 4 summarizes results from the Monte Carlo simulations in which minimal uncertainties for the ingestion of risk coefficients were quantified using either 90%, 80%, or 50% credible intervals. The 90% credible intervals were the intervals that encompass 90% of the simulated risk coefficients between Q_5 and Q_{95} (where Q_5 is the 5% sample quantile of the risk coefficients, and Q_{95} is the 95% quantile). The 80% and 50% credible intervals were obtained using Q_{10}, Q_{90}, and Q_{25} and Q_{75} respectively. The main results of our analysis are summarized in the first three columns. For about 50% of the radionuclides the ratio of Q_{95}/Q_5 was less than 23. The ratio Q_{75}/Q_{25} was much smaller; for about 50% of

the radionuclides – not necessarily the same ones – the ratio of the midrange values was less than 3.4. By taking the square roots of the ratios in the first columns, one may conclude that subject to the limitations of this analysis, the accuracy of most of the risk coefficients ranges from a factor of about 4 (rounded square root of 12.3) to about 25 (about the square root of 540). (All values in the interval from Q_5 to Q_{95} are within a factor of roughly $(Q_{95}/Q_5)^{1/2}$ of the risk coefficient, provided the risk coefficient is near the geometric mean of Q_5 and Q_{95}.)

Table 4:
Quantiles for the ratios of upper and lower limits of subjective uncertainty intervals, obtained using Monte Carlo simulations, for cancer risk coefficients for ingestion.

% of radionuclides with smaller ratios of upper to lower limits	Q95/Q5	Q90/Q10	Q75/Q25
5	11.5	6.6	2.6
20	15.6	8.2	2.9
40	20	9.8	3.2
50	23	11.0	3.4
60	26	12.3	3.6
80	49	19.6	4.6
95	562	104	10.4

As part of the analysis, an assessment was made of the relative contribution of each of the different sources of uncertainty. For this analysis, the uncertainties were categorized as to whether they relate to 1) models for the calculation of absorbed dose, 2) radiogenic cancer risk models for low-LET radiation at high dose and high dose rate, or 3) the dose modifiers RBE and DDREF. This was accomplished by comparing a) the variance of the log transformed risk values generated when factors associated with all but one source of uncertainty type of model were varied with b) the variance of the transformed risk values when factors associated with each of the sources were varied simultaneously.

A particular source of uncertainty was considered to be dominant if its contribution accounted for more than half of the variance of the log transformed risk models. For 221 out of 758 radionuclides the dominant source of uncertainty was "absorbed dose", and for 483 radionuclides the dominant source was the "risk model". For the remaining 54 radionuclides none of the three sources of uncertainty dominated. The term "absorbed dose" refers to uncertainties relating to the use of both biokinetic and dosimetric models for estimating the absorbed doses for each tissue type. The biokinetic models characterize the biokinetics of a radionuclide in the lungs and gastrointestinal tract and its absorption to blood, as well as its systemic biokinetics. Dosimetric models relate to the conversion of activity distributed in the human body to absorbed dose to tissues. The term "risk model" includes only the uncertainties relating to the assessment of risk per unit absorbed dose for low LET radiation at high doses/rates (and therefore does not include uncertainties relating to DDREF or RBE). It should be noted that for ingestion, there was no radionuclide for which the dominant source of uncertainty relates to the absorbed dose modifiers RBE and DDREF. Uncertainty tends to be smallest for radionuclides for which the dominant source of uncertainty is the "risk model" and greatest when the dominant source is associated with determination of absorbed dose.

Discussion

The uncertainties summarized in the previous section were based on a simplified model in which the risk per unit activity for each type of radiation (high-LET or low-LET) is expressed as the product of three components: first, the risk per absorbed dose received by target tissues (for low-LET high dose and dose rate radiation); second, modifying factors applied to the first component to account for type of radiation, dose, and dose rate; and finally the absorbed dose per unit activity. This formulation allows a convenient allocation of uncertainties associated with the models used to derive the risk coefficients, and is a logical extension of formulations in previous evaluations of uncertainties in risks from whole-body irradiation (NCRP 1997; EPA 1999).

Results from this uncertainty analysis need to placed in perspective, since it is true that subjective judgment played a role in almost every step of the process used to generate the simulations. We did not attempt to evaluate uncertainties relating to the validity of the linear-no-threshold hypothesis, since this simply is not feasible. As discussed earlier, we based our analysis on a simplified risk model, which did not account for age-dependencies in either absorbed doses or risks per absorbed doses. It follows that uncertainties for radionuclides that concentrate in bones (for long periods of time) may be understated in this report.

With these limitations in mind, we nevertheless hope that this report provides a reasonable evaluation of the uncertainties for the ingestion of radionuclides in FGR13.

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Effects of Baseline on Uncertainty of Radiation Risk Models

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Abstract

ICRP, BEIR, UNSCEAR, and EPA developed the radiation risk projection models, which are based on the epidemiological data especially of Hiroshima-Nagasaki atomic bomb survivors. To apply the data to the other population, cancer mortality data and survival data are used as the baseline. The purpose of this study is to examine the effects of baseline on the radiation risk projection models. At first, using the multiplicative risk projection model, we consider whether or not the ICRP's risks are statistically significant in the present. For Japan, there exist the significant differences in most of cancer sites except for esophagus and leukemia. For the USA, there are a fewer sites where the difference is more significant than Japan. In Japan, the years that the risk on a year is effective in the future are only one year in colon and total cancers *etc.*, and a few years in most of the other cancer sites. By extrapolating cancer mortality, we predict the risks in the future. Also, using the excess relative risk based on attained age, which are included in the radiation risk projection model, the effects of baseline are examined.

Introduction

Using the radiation risk projection model, we can estimate the lifetime excess cancer mortality risk in a certain population, where the excess relative risk (ERR) coefficient obtained by the epidemiological study, mainly of Japanese atomic bomb survivors, is used. Then, to take a difference between the population into consideration, spontaneous cancer mortality data and survival data in a population are applied as the baseline. NCRP (1997) and EPA (1999) evaluate the uncertainties in the radiation risk projection model by assuming the statistical distributions to the uncertain sources, which are dose and dose rate effectiveness factor (DDREF), population transfer, epidemiology, error in the death diagnosis, dosimetry and so on. In uncertainty analyses of both organizations, though the assumed distributions are different, the DDREF has the largest contribution. On the other hand, the population transfer is a source that the order of the contribution is greatly different between two organization's results, that is, it means that the contribution of uncertainty varies greatly according to the distribution for the baseline.

ICRP (1991) derives the lifetime excess cancer mortality risks by averaging the values calculated from each baseline data of five countries including Japan (mortality data in 1978 and survival data in 1986-1987) and USA (mortality in 1973-1977 and survival in 1985), whose details are given by Land and Sinclair (1991). However, since these mortality data are the older data than twenty years, we wonder whether the risks projected by ICRP are available in the present.

Let e be the age at exposure and D an exposure dose. For a cancer site i, suppose that $M_i(x)$ denotes the mortality rate at age x, $S(x)$ denotes the proportion of survival at age x, and $ERR_i(e,D)$ denotes the ERR given by the exposure age and dose. Then, the multiplicative risk projection model is expressed as:

$$u_i(e,D) = ERR_i(e,D)\int_{e+L}^{e+L+P} M_i(x)\frac{S(x,D)}{S(e)}dx, \qquad \textbf{(1)}$$

where the minimum latency time L is 2 if leukemia, 10 if else and the plateau period P is 40 if leukemia, positive infinity if else. Risk for total cancer is obtained by summing up $u_i(e,D)$ for all i. To exclude the uncertain effect by DDREF and simplify the projection, we set the acute exposure dose 1 Sv. Then, the model is transformed such as follows:

$$u_i(e) = ERR_i(e)\int_{e+L}^{e+L+P} M_i(x)\frac{S(x)}{S(e)}dx. \qquad \textbf{(2)}$$

Based on the age at exposure, the ERR is estimated by the epidemiological study. However, the ERR based on the attained age are recently proposed by Kellerer and Barclay (1992), and Pierce and Mendelsohn (1999), which state that the ERR based on the attained age is more fit to the data of atomic bomb survivors than the one based on the age at exposure. Therefore, we apply the ERR based on the attained age to the risk projection model. Then, the model is given as the function of the age at exposure:e and the attained age:a, and expressed by:

$$u_i(e,a) = ERR_i(a)\int_{e+L}^{e+L+P} M_i(x)\frac{S(x)}{S(e)}dx. \qquad \textbf{(3)}$$

In this study, following to Land and Sinclair (1991), we deal with esophagus, stomach, colon, lung, female breast, ovary, bladder, leukemia and residuals as target cancer sites. Also, Six kinds of ages (0, 10, 20, 30, 40, 50) are used as the age at exposure.

This paper is organized as follows. In the following section, using the test of equality, we examine whether or not the ICRP's risks are statistically significant in the present. In the next section, the years that the baseline data is effective are studied. In the subsequent section, the risks in future are projected by the extrapolation of the baseline. In the next section, using the ERR based on the attained age, the same significance as the above is examined.

Tests of the Equality for Risks

In this section, from the statistical viewpoint, we examine whether or not Japanese and USA's risks given by ICRP (1991), whose baseline data are given by Lang and Sinclair (1991, Table 2, 3), are effective in the present, respectively. As the latest baseline data, we can get Japanese mortality in 1999[7], Japanese survival in 1999 from the homepage of Ministry of Health, Labour and Welfare, and USA's baselines in 1998 from the homepage of National Center for Health Statistics.

Let the index *c* be the country; *J* (Japan) or *U* (USA), the index *s* be the sex; *M* (male) or *F* (female), and the index *y* be the data year or *ICRP* (data used by ICRP). The risk is projected using the equation (2), and is expressed by $u_i(e;c,s,y)$. That is, for a site *an exposure age e, the risk obtained by using the Japanese male's baseline data in 1999 and I* is expressed by $u_i(e;J,M,1999)$. Then, for each sex, by comparing $u_i(e;J,*,1999)$ with $u_i(e;J,*,ICRP)$, and $u_i(e;U,*,1998)$ with $u_i(e;U,*,ICRP)$, the effectiveness of the ICRP's risks in the present is statistically examined.

It is assumed that the number of cancer-site-specified death and the number of survival are independent random variables, each of which follows a binomial distribution, respectively. By iterating that we generate the random numbers according to these assumptions and calculate the risk projection model, the distributions of the risks are investigated. In this case, the iteration is done 5000 times. Then, by illustrating the histogram or the Q-Q plot, it seems that each risk has normal distribution. Therefore, we may use the test of the equality. Since we can use the same test regardless of the site, the exposure age, the country and the sex, we explain the case of Japanese male for a cancer site and an exposure age.

Let $U_i(e;J,M,ICRP)$ and $U_i(e;J,M,1999)$ be a random variable independently distributed as normal with the means m_{ICRP}, m_{1999} and the variances s^2_{ICRP}, $s^2_{1999,}$ respectably. The null hypothesis of the testing problem is expressed as $m_{ICRP} = m_{1999}$. Then, for a site *an exposure age e, and I* the test statistic is given by

$$Z_i(e) = \frac{U_i(e,J,M,ICRP) - U_i(e,J,M,1999)}{\sqrt{s^2_{ICRP} + s^2_{1999}}}, \qquad \textbf{(4)}$$

which is distributed as a normal with the mean 0 and the variance 1. Since we may regard $u_i(e;J,M,1999)$ as the risk of population mean in Japanese male of 1999 under a specific condition, which is sufficient for the large population, this value can be calculated by substituting u_i for U_i. Then, by the normality of Z_i, the probability that the null hypothesis is rejected, which is called as *p*-value, is obtained. Here, we assess the testing problem by the significance level of 0.05. That is, when the *p*-value is below 0.05, it means that the null hypothesis is rejected and that the risk given by ICRP (1991) is significantly different from the risk basing on the baseline data in 1999. The *p*-values for Japan and USA are shown in Tables 1 and 2, respectively.

In case of Japan (Table 1), except for all ages of esophagus and for most ages of leukemia, it is seen that there exist the significant differences between two risks and that most ICRP's risks are statistically not effective in the present. Since this result depends on the differences of the baseline data, we may say that the baseline affects the risk projection model very much. In case of USA (Table 2), though there are more sites that the difference between two risks is not significant than Japan, especially for female, the ICRP's risks in some sites are statistically not effective in the present. So, for the risk projection model, we examine the years that the Japanese baseline data is applicable in future.

Table 1:
The p-values of the statistic in Japan, classified by sex, site and exposure age.

When the p-value < 0.05, there exists the significant difference between $u_i(e;J,*,ICRP)$ and $u_i(e;J,*,1999)$

Sex	Exposure Age	0	10	20	30	40	50
M	Esophagus	0.221	0.236	0.242	0.237	0.246	0.474
	Stomach	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01
	Colon	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01
	Lung	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01
	Bladder	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01
	Leukemia	0.086	0.271	0.585	0.674	0.044	< 0.01
	Residual	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01
	Total	***< 0.01***	***< 0.01***	***< 0.01***	***< 0.01***	***< 0.01***	***< 0.01***
F	Esophagus	0.192	0.189	0.189	0.186	0.170	0.158
	Stomach	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01
	Colon	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01
	Lung	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01
	Breast	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01
	Ovary	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01
	Bladder	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01
	Leukemia	0.098	0.230	0.478	0.920	0.144	< 0.01
	Residual	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01
	Total	***< 0.01***	***< 0.01***	***< 0.01***	***< 0.01***	***< 0.01***	***< 0.01***

Table 2:
The p-values of the statistic in USA, classified by sex, site and exposure age.

When the p-*value < 0.05, there exists the significant difference between* u_i(e;U,*,ICRP*) and* u_i(e;U,*,1998*)*

Sex	Exposure Age	0	10	20	30	40	50
M	Esophagus	0.016	0.019	0.019	0.020	0.020	0.014
	Stomach	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01
	Colon	0.265	0.223	0.214	0.202	0.232	0.302
	Lung	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01
	Bladder	0.748	0.698	0.688	0.665	0.652	0.740
	Leukemia	0.127	0.417	0.775	0.233	< 0.01	< 0.01
	Residual	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01
	Total	***0.999***	***< 0.01***	***< 0.01***	***< 0.01***	***< 0.01***	***< 0.01***
F	Esophagus	0.686	0.699	0.700	0.700	0.642	0.441
	Stomach	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01
	Colon	< 0.01	< 0.01	< 0.01	< 0.01	0.011	0.035
	Lung	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01
	Breast	0.164	0.143	0.146	0.186	0.421	0.762
	Ovary	0.674	0.659	0.678	0.736	0.936	0.352
	Bladder	0.386	0.373	0.371	0.369	0.369	0.419
	Leukemia	0.179	0.370	0.537	0.969	0.193	< 0.01
	Residual	0.394	0.433	0.423	0.398	0.260	0.075
	Total	***< 0.01***	***< 0.01***	***0.715***	***< 0.01***	***< 0.01***	***< 0.01***

Effective years of baseline

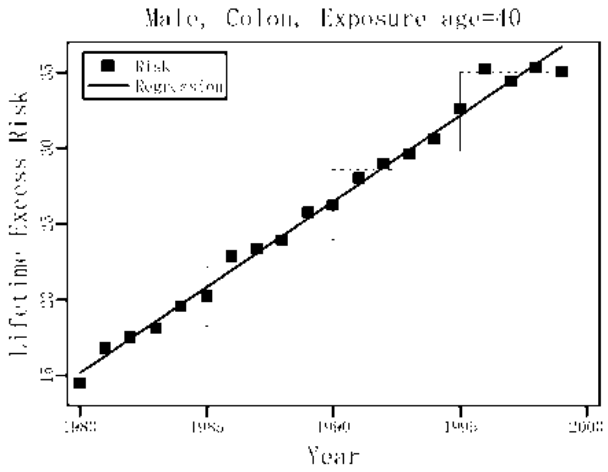
In this section, using the Japanese baseline data from 1980 to 1999 every one-year, the years that the baseline is trustworthy and available in future is examined in view of the lifetime excess cancer mortality risk, which depends on the baseline. At first, let 1985, 1990 and 1995 years be three representative points. For each point, by ordering the values obtained by the same simulation as the previous section (the iteration times is 2000), we can obtain the boundary value deciding the 95% confidence intervals (CI) on each representative point. Also, we consider the linear regression models for the risks calculated on every one-year such as:

$$y = \beta_0, \qquad y = \beta_0 + \beta_1 x,$$
$$y = \beta_0 + \beta_1 x + \beta_2 x^2, \qquad y = \beta_0 + \beta_1 x + \beta_2 x^2 + \beta_3 x^3,$$

where x and y denote the data year and the risk, respectively. The parameters for each model are estimated by the regression analysis. By the C_p criterion (Mallows (1973), which is one of the methods to select statistically the fittest regression model, one model of them is selected and the degree of regression model is 2 or 3 for most sites and exposure ages. Then, we can consider the effectiveness of the risk by the 95% points and the fittest regression model. Three examples are shown in Figures 1, 2 and 3. Since Figure 1 shows that the risks based on data of 1985, 1990 and 1995 are available for one year or two years, we conclude that, in the meaning of "at least", the risk for colon cancer of a Japanese male exposed at age 40 is effective for one year. Similarly, Figure 2 shows that the risks based

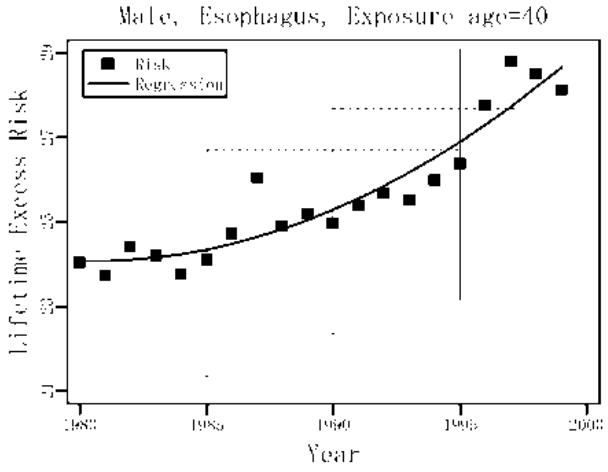
on data of 1985, 1990 and 1995 are available for nine years, six years and more than four years, respectively. Therefore, we conclude that the risk for esophagus cancer of a Japanese male exposed at age 40 is effective for four to six years. By Figure 3, the risk for esophagus cancer of a Japanese female exposed at age 40 is effective for more than four years. The conclusion is summarized in Table 3. For example, in the case of stomach cancer, when we project the risk using the baseline data in 2000, this means that its risk is effective until 2002 or 2003. As a whole, the effective years in future are a few in most sites. Therefore, we can say that the present risk projection model is affected by the baseline.

Figure 1:
The lifetime excess cancer mortality risks for colon cancer of Japanese male exposed at age 40 and the fittest regression model.



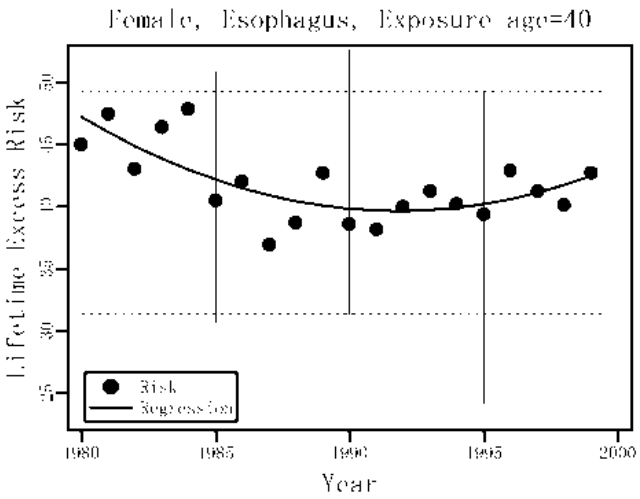
The x- and y-axes denote the year of the baseline data and the risk per 10,000 persons, respectively. The squares are the risks obtained by the baseline on every one-year. The line is the fittest model selected by the C_p criterion. The vertical lines in 1985, 1990 and 1995 denote the 95% CI. In case of the risk in 1995, since the 95% CI intersects with the regression model by 1998 (dashed line), we can express that the risk in 1995 is effective for two years in future. Similarly, both risks in 1985 and 1990 are effective for one year.

Figure 2:
The lifetime excess cancer mortality risks for esophagus of Japanese male exposed at age 40 and the fittest regression model.



The risks in 1985, 1990 and 1995 are effective for nine, six and more than four years, respectively. Therefore, we conclude that this risk is effective for four to six years.

Figure 3:
The lifetime excess cancer mortality risks for esophagus of Japanese female exposed at age 40 and the fittest regression model.



All risks in 1985, 1990 and 1995 are included in 95% CI.
Therefore, we conclude that this risk is effective for more than four years.

Table 3:
Effective years of the baseline in future, which is given regardless of the age at exposure.

Site (Sex)	Years
Total(M,F), Colon(M,F), Lung(M), Residual(M)	0~1
Stomach(M,F), Bladder(M), Lung(F), Breast(F), Residual(F)	2~3
Esophagus(M), Ovary(F), Bladder(F)	4~6
Esophagus(F), Leukemia(M,F)	4~

Future risk by extrapolation of baseline

As described in the previous section, in most cancer sites, the years that the baseline data is effective in the future is not so long. So, we predict the risk in the future by extrapolating cancer mortality.

For a site, a sex and an age group, by applying the simple linear regression ($Y=A+Bt$) to cancer mortality data from 1980 to 1999 every one-year, and extrapolating its regression, cancer mortality in the future is predicted. Here, the exponential regression ($Y=Ae^{Bt}$) is applied when the mortality decreases sharply. Then, the risk in future can be obtained by applying the extrapolated cancer mortality to the risk projection model (2). Two examples that the risks in 2005 and 2010 are predicted are shown.

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